Where do we go from here? M*A*S*H is over; we have lost a friend. Perhaps something else can fill the self-reflective void we now might have. Perhaps in the 11 years since its start, we have learned how to do that self-reflection ourselves.

To the 4077th M*A*S*H: Goodbye, farewell, amen.

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Hypervitaminosis A, Hypercalcemia and Hemodialysis

TO THE EDITOR: I wish to clarify two statements, and to give additional information, concerning the recent report by Baxi and Dailey on "Hypervitaminosis A: A Cause of Hypercalcemia."[1]

The authors state, "Hypercalcemia has been reported only rarely [in relation to hypervitaminosis A] and approximately seven patients have been described previously." However, at least 6 additional cases (a total of 13 or more) were linked to vitamin A toxicity by 1978 (examples are cited in references[2-4]). Hypercalcemia is actually reported in roughly a quarter to a third of all cases, at least in adolescents and adults, and it could be considered a fairly common symptom. I agree with Baxi and Dailey, however, that hypervitaminosis A may be too rarely *considered* as a cause of hypercalcemia, and their report is valuable to emphasize this point.

The authors' statement that hypervitaminosis A "has led to fatal portal hypertension on occasion. . . ." is not well founded. No fatalities are mentioned in the authors' references for this statement. In fact, in my study of the literature,[5] I have found *only one death* ever attributed to vitamin A—an English PhD chemist whose cause of death was stated to be liver cirrhosis, without mention of portal hypertension.[6] Despite illness and medical warnings, he took supplements of more than a million IU daily for long periods. Polar bear liver is well known to sometimes cause brief, acute symptoms, but apparently without reported fatalities.[5]

It is not clear how vitamin A has acquired its lethal reputation. Other nutrients with lesser reputations for toxicity have caused far more fatalities; for example, there have been perhaps a score of deaths in the United States from vitamin D, and hundreds from iron supplements ingested by children.

An additional valuable report concerns patients undergoing hemodialysis. They seem to be unusually sensitive to vitamin A supplements and prone to hypercalcemia which abates when supplements are withdrawn.[7] This report, like that of Baxi and Dailey, presents information which can help physicians prevent and correctly diagnose vitamin A toxicity.

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REFERENCES

1. Baxi SC, Dailey GE III: Hypervitaminosis A: A cause of hypercalcemia. West J Med 1982 Nov; 137:429-431
2. Fisher G, Skillern PC: Hypercalcemia due to hypervitaminosis A. JAMA 1974; 227(12):1413-1414
3. Hofman KJ, Milne FJ, Schmidt C: Acne, hypervitaminosis A and hypercalcemia. S Afr Med J 1978; 54(14):579-580
4. Breslaw RC: Hypervitaminosis A. Arch Pediat 1957 Apr; 74:139-152
5. Davis DR: Using vitamin A safely. Osteopathic Med 1978 Oct; 3: 31-34
6. Leitner ZA, Moore T, Sharman IM: Fatal self-medication with retinol and carrot juice. Proc Nutr Soc 1975 Sep; 34:44A-45A
7. Farrington K, Miller P, Varghese Z, et al: Vitamin A toxicity and hypercalcaemia in chronic renal failure. Br Med J 1981; 282(6281): 1999-2002

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Drs Dailey and Baxi Reply

TO THE EDITOR: We wish to thank Dr Davis for his comments and interest in our case report. We appreciate his pointing out additional instances of reported hypercalcemia with vitamin A intoxication not cited in our references. The frequency of hypercalcemia is difficult to state with certainty; this clearly does underscore the need to consider it as a cause of readily reversible hypercalcemia.

Dr Davis is also correct in stating that death did not occur in the instance of cirrhosis and portal hypertension cited in our references. This was cited principally as the best histologic verification of this finding. Reference 6 in Dr Davis' letter is perhaps the best documented death in humans. Certainly, a variety of hepatic lesions have been reported in adults as well as children. While documented deaths have also occurred in laboratory animals, the doses and duration of therapy necessary to produce the lesions remain open to question.

The purpose of our case report was certainly not to denigrate the importance of vitamin A and other retinols in human nutrition and its potential importance in prevention of other serious illnesses. However, there remains no clear documentation of benefit in prescribing amounts of vitamin A far in excess of the current recommended daily allowances. We continue to feel that the use of pharmacologic doses of potentially toxic substances, especially fat soluble vitamins, should be more carefully controlled and available through prescription only.

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